

# Student Paper Communication étudiante

## Is there a link between pollutant exposure and emerging infectious disease?

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**Abstract** — A scoping literature review found evidence supporting the hypothesis that a population's pollution status could help refine classification of emerging infectious disease (EID) hotspots. Systematic literature reviews and studies designed to specifically test the predictive value of pollutant status on EID risk are recommended.

**Résumé** — Y a-t-il un lien entre l'exposition aux polluants et les maladies infectieuses émergentes? Une recension extensive de la littérature a permis de trouver des données probantes appuyant l'hypothèse que l'état de pollution de la population pourrait aider à raffiner la classification des points chauds des maladies infectieuses émergentes (MIE). Des examens systématiques de la littérature et des études conçues spécifiquement pour tester la valeur prédictive de l'état des polluants sur le risque des MIE sont recommandés.

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**E**merging infectious diseases (EIDs) remain an ongoing threat to human and animal health. Global vulnerability to these diseases is not homogeneous, suggesting that there are specific factors affecting the susceptibility of a population to EIDs (1). There is growing interest in being able to detect populations and locations vulnerable to EIDs. Vulnerability to EIDs is influenced by the extent of exposure to the pathogen, susceptibility to that pathogen, and the ability to cope with the infection. Significant research effort has been focused on exposure to pathogens, finding both new pathogens and new exposure routes. In this paper, we set out to determine if there is evidence supporting the hypothesis that pollution can affect population susceptibility to infection, and thus understanding if a population's pollution status could help refine EID hotspot classifications.

A scoping literature review was performed (2). Scoping reviews are undertaken to determine the need or value of conducting a systematic literature review (3). English language peer-reviewed papers from 1980 to the present that contained key words [including infection, disease, pollution, polychlorinated biphenyls (PCBs), pesticide, heavy metal] were sought using search engines (PubMed, Web of Science). Only field studies, studies of natural experiments, or accidental exposures that drew a link from pollutant exposure to an infectious disease outcome were included in the results (Table 1). No studies designed to explicitly test the hypothesis that pollution status can affect

EID risk were found. Most papers with the key words “emerging infectious disease” and “pollution” focused on concepts of microbial pollution rather than chemical pollutants. Therefore, we began examining associations between infectious disease and pollution status. Of the 23 papers that met our inclusion criteria, 11 species and 4 contaminant groups were examined. Most were cohort studies (61%); other study types included case-control (17%), cross-sectional (13%), case-crossover (4%), and time series (4%).

Simple infectious disease models predict that immunocompromising stressors which do not affect host abundance will increase disease (4). Previous work has established that pollutants such as PCBs and organochlorines are immunotoxic (5). Field studies and opportunistic case studies from accidental exposures showed that animals and humans had different infectious disease responses associated with their pollution status. The cases summarized in Table 1 establish plausible associations between pollution and infections across taxa, for different pollutants and different pathogens. Different study types found similar associations (Table 1). Both endemic infections and disease outbreaks were associated with pollution status, as was the severity of some infections.

The presence of a plausible biological mechanism, the observation of a similar effect across locations, species, and pollutants, support our hypothesis that pollution status may be an indicator or determinant of susceptibility to infectious disease. An alternative hypothesis is that both infection status and pollution status reflect environmental milieu and social circumstances. Exposure to environmental risk factors is unequally distributed and is influenced by social conditions. The associations between pollution and infections observed in this review could reflect spatial co-occurrence of risk factors rather than causal relationships. However, the observation of these relationships in cohort and case-control studies and in animals and humans

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**Table 1.** Summary of field and case studies examining the relationships between contaminants and infectious disease outcomes in humans and animals

Contaminant		Species	Infectious disease outcome	Reference
Polychlorinated biphenyls (PCBs)	N/A	Human ( <i>Homo sapiens</i> )	Increased number and type of infectious diseases in breast-fed infants of mothers who consumed contaminated fish	(8)
		Human ( <i>Homo sapiens</i> )	Increased frequency of bronchitis and influenza at 6 mo, increased frequency of influenza and otitis media at 6 y	(9)
		Human ( <i>Homo sapiens</i> )	Higher incidence of viral infections (colds) in infants born to occupationally exposed mothers	(10)
		Harbor porpoise ( <i>Phocoena phocoena</i> )	Increased mortality due to infectious disease	(11)
		Harbor porpoise ( <i>Phocoena phocoena</i> )	Increased mortality due to bacterial infections	(12)
		Striped dolphins ( <i>Stenella coeruleoalba</i> )	Increased incidence of morbillivirus	(13)
Pesticides	Organochlorines	Human ( <i>Homo sapiens</i> )	Increased incidence of otitis media in breast-fed infants	(5)
		Human ( <i>Homo sapiens</i> )	Increased frequency of respiratory tract infections	(14)
		Human ( <i>Homo sapiens</i> )	Increased incidence of <i>Herpes zoster</i>	(15)
		Common seals ( <i>Phoca vitulina</i> )	Correlation with phocine distemper (morbillivirus) epizootic	(16)
	Organochlorines (including DDT) + PCBs	Glaucous gulls ( <i>Larus hyperboreus</i> )	Increased incidence of parasitic nematodes	(17)
	Organochlorines (including DDT) + PCBs + polybrominated diphenyl ethers	Burbot ( <i>Lota lota</i> )	<i>Mycobacterium salmoniphilum</i> infection in addition to other gross and microscopic pathologies	(18)
	Neonicotinoid (Imidacloprid, Thiacloprid)	Honeybee ( <i>Apis mellifera</i> )	Correlation with infection with <i>Nosema microsporidians</i>	(19–21)
	Agricultural runoff	Wood frogs ( <i>Rana sylvatica</i> )	Correlation with infection with <i>Ribeiroia</i> sp. trematodes	(22)
Air pollution	N/A	Human ( <i>Homo sapiens</i> )	Increased frequency of respiratory infections in children	(23)
		Human ( <i>Homo sapiens</i> )	Correlation between hospitalizations for respiratory infections in children and coarse particulate matter	(24)
		Human ( <i>Homo sapiens</i> )	Correlation between air pollution and general practice consults for respiratory infections	(25)
Heavy metals	Lead	House sparrow ( <i>Passer domesticus</i> )	Association between <i>Plasmodium relictum</i> infection and lead levels	(26)
	Selenium	Human ( <i>Homo sapiens</i> )	Significant concentrations of heavy metals including selenium found in AH1N1 patients	(27)
	Arsenic	Human ( <i>Homo sapiens</i> )	Increased mortality from bronchiectasis due to early-life exposure	(28)
		Human ( <i>Homo sapiens</i> )	Increased incidence of lower respiratory tract infection and diarrhea in infants of mothers exposed to arsenic	(29)

A scoping literature review with specific inclusion criteria was performed, and the results yielded 23 articles including studies on 4 categories of contaminants and 11 species. Articles using **cohort**, **case control**, **cross-sectional**, case-crossover, and **time series** study designs were found. Most articles (61%) were cohort studies. N/A — Not applicable.

living in different ecological settings reduces the likelihood of this alternative hypothesis.

The scoping methodology allowed us to undertake a general scan for study design and relevance. The results suggest that a detailed, and critical systematic review of methods and results across taxa, pollutants, and situations is warranted. Our goal was not to prove the relationship between pollution and EID risk, but rather to determine if this was a reasonable hypothesis to pursue; as such, we sought papers that established a positive link between pollution and infectious disease introducing a confirmation bias.

The co-occurrence of pollutants and infections argue in favor of public health and animal health programs that aim to increase capacity to cope with environmental stressors in general rather than a hazard-by-hazard approach. Distribution maps for historical PCB emission ([www.nilu.no/projects/globalpcb/](http://www.nilu.no/projects/globalpcb/)), satellite-based measurement of aerosolized fine particulate matter (6), and statistical modelling of geogenic groundwater arsenic contamination (7) show information regarding worldwide pollution which could be used in correlation studies and potentially EID prevention effort allocation.

Health professions need to focus on reducing population vulnerability and increasing adaptive capacity when planning for large environmental threats such as climate change. This paper suggests a similar approach may be warranted for locations suffering from co-occurrence of pollution and infections. Another argument to reduce the world's pollution hardly seems necessary; however, primary prevention programs of EID could include reducing environmental contamination. Through preventing EID outbreaks and spread, human, animal, emotional, and monetary costs could be decreased. A systematic literature review and future studies designed specifically to examine the effects of pollutants on EID vulnerability are recommended.

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